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Javanainen, Tuija

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Prognostic impact of baseline and residual SYNTAX scores in cardiogenic shock

Tuija Javanainen MD¹  | Jordi Sans-Roselló MD² | Veli-Pekka Harjola Assoc. Prof.³ | Markku S. Nieminen Prof.¹ | Johan Lassus Assoc. Prof.¹ | Alessandro Sionis Assoc. Prof.² | Marjut Varpula PhD¹ | Raija Jurkko PhD¹

¹Cardiology, University of Helsinki, Heart and Lung Center, Helsinki University Hospital, Helsinki, Finland

²Department of Cardiology, Acute and Intensive Cardiovascular Care Unit, Hospital de la Santa Creu i Sant Pau, Biomedical Research Institute IIB-Sant Pau, Universitat Autònoma de Barcelona, CIBER-CV, Barcelona, Spain

³Emergency Medicine, Department of Emergency Medicine and Services, University of Helsinki, Helsinki University Hospital, Helsinki, Finland

Correspondence

Tuija Javanainen, MD, Tallbergin puistotie 7B26, 00200 Helsinki, Finland.
Email: tuija.javanainen@outlook.com

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Abstract

Objectives: The aim was to assess the extent of coronary artery disease and revascularization using baseline SYNTAX Score (bSS) and residual SYNTAX Score (rSS) in patients with cardiogenic shock (CS) secondary to ST-segment elevation myocardial infarction (STEMI). The prognostic impact of SYNTAX Score (SS) was evaluated and assessed for additive value over clinical risk scores.

Background: bSS and rSS have been proven to be useful in risk stratification in stable coronary artery disease as well as in acute coronary syndromes, but they have not been studied in STEMI related CS.

Methods: Patients from a multinational prospective study of CS were analyzed. The study population was divided into tertiles according to bSS. The Cox regression and receiver operating characteristic (ROC) curves were used to assess the predictive power of SS.

Results: Of the 61 studied patients, 85% were male and the mean age was 67 years. Median bSS was 22 (15–32) and rSS 7 (0–13). Ninety-day mortality was 43%. bSS had negative prognostic value in multivariable analysis (HR 1.06, 95% CI 1.01–1.10). However, additive value over clinical risk scores was limited. rSS was not associated with mortality, whereas post-percutaneous coronary intervention (PCI) TIMI flow 3 of infarct-related artery (IRA) predicted better survival.

Conclusions: In STEMI related CS, the added value of bSS and rSS over clinical assessment and risk scores is limited. Our results suggest that while immediate PCI in order to restore blood flow to the IRA is essential, deferring the treatment of residual lesions does not seem to be associated with worse prognosis.

KEYWORDS

acute coronary syndrome, prognosis, revascularization, shock

1 | INTRODUCTION

SYNTAX Score (SS) is an angiographic scoring system that measures the burden of coronary artery disease and has been shown to be an independent predictor of cardiovascular events and mortality in stable coronary artery disease¹ as well as in acute coronary syndromes (ACS).^{2–6} In the current literature, there are no data describing the prognostic value of baseline SS (bSS) in ST-segment elevation

myocardial infarction (STEMI) related cardiogenic shock (CS). Residual SS (rSS) measures the extent of coronary disease after percutaneous coronary intervention (PCI) and can be used as an indicator of completeness of revascularization. Current guidelines suggest multivessel PCI in CS,⁷ but new data about revascularization strategies in CS have been published after these recommendations: the CULPRIT-SHOCK trial showed worse outcome in CS with multivessel PCI in comparison with revascularization of culprit artery only.⁸ Previously, the predictive

value of rSS has been shown in stable coronary artery disease,^{9–11} as well as in patients with ACS,^{12–14} but not studied in the context of CS.

The aim of this study was to evaluate the extent of coronary artery disease and success of revascularization in STEMI-related CS patients treated with PCI. Second, the predictive value of bSS and rSS in CS patients was evaluated over clinical and angiographic values, including TIMI flow grade of the infarct-related artery (IRA). Finally, we assessed the additive prognostic value of SS over three clinical risk scores: CardShock risk score,¹⁵ IABP-SHOCK II risk score¹⁶ and GRACE risk score.¹⁷

2 | MATERIALS AND METHODS

2.1 | Study population

This is a sub-analysis of the CardShock study, a prospective observational multicenter investigation of the prognosis and treatment of CS. The CardShock study was conducted from October 2010 to December 2012 in eight European countries in nine hospitals. The design, methodology and primary results have been published before.¹⁵ The study was approved by local ethics committees and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each patient or a next of kin.

Briefly, adult patients who fulfilled predefined criteria for CS were included within 6 hr from the detection of CS. The criteria for shock were systolic blood pressure less than 90 mmHg for at least 30 minutes despite adequate fluid therapy or need for vasoactive therapy, and ≥ 1 signs of inadequate organ perfusion: confusion or altered mental status, cool extremities, oliguria less than 0.5 ml/kg/h for the previous 6 hr, or blood lactate greater than 2 mmol/l. Patients with ongoing hemodynamically significant arrhythmia, or shock after cardiac or non-cardiac surgery were excluded.

Patients' demographics, medical history and clinical characteristics were collected. Hemodynamic parameters and laboratory measurements were registered. Arterial blood lactate and pH were analyzed locally and creatinine, high-sensitivity troponin T (hs-TnT), and N-terminal pro-B-type natriuretic peptide (NT-proBNP) (Roche Diagnostics, Basel, Switzerland) were analyzed centrally. Estimated glomerular filtration rate (eGFR) was calculated from creatinine values using the CKD-EPI Creatinine equation. Echocardiography was performed at study entry. The patients were treated according to local practice and given treatments were registered.

For the present substudy, angiographic images from the two largest centers (Helsinki and Barcelona) were analyzed and combined with the clinical data from the CardShock database. STEMI patients treated with primary PCI or rescue PCI were included. STEMI was defined according to the third universal definition of myocardial infarction.¹⁸ Exclusion criteria were previous coronary artery bypass grafting (CABG), emergency CABG after angiography, or mechanical complication of STEMI as an etiology of CS. The primary endpoint was all-cause mortality at 90-day follow-up. The vital status was confirmed by the patient or the next of kin, or through hospital or population registers.

2.2 | SYNTAX scores

SS was measured from angiographic images using the SS algorithm.¹⁹ Two experienced cardiologists (MV and JS) analyzed the angiograms post hoc blinded to patient data. The interobserver variability (tertil partitioning) for the SS examiners was calculated in 48 independently analyzed angiograms: Cohen's Kappa statistics was 0.59 (95% CI 0.40–0.78, $P < 0.01$) for bSS and 0.65 (95% CI 0.32–0.97, $P < 0.001$) for rSS, signifying moderate interobserver reliability comparable to the SYNTAX trial¹ and also to the previous studies with ACS patients.^{3,5,21}

All lesions with stenosis greater than 50% in vessels with diameter of more than 1.5 mm were scored. SS was calculated at three time points:

1. Baseline SS1 (bSS1) was measured from initial diagnostic angiogram. Completely occluded IRA with TIMI flow 0 or 1 was scored as a total occlusion.
2. Baseline SS2 (bSS2) was measured after wiring or thrombectomy, if TIMI flow of IRA improved and severity of the lesions could be measured downstream. If TIMI flow did not improve or anatomy of IRA could not be assessed, bSS2 was the same as bSS1.
3. Residual SS was measured after primary PCI was completed. If further angiograms were performed during the hospital stay, the rSS was measured after all PCI procedures.

2.3 | Statistical analysis

The continuous data are presented as means and standard deviations (SD) or as medians and interquartile ranges (IQR). The study population was divided into tertiles by bSS1. Differences between tertiles were analyzed with one-way ANOVA, the Kruskal-Wallis or logistic regression as appropriate. The changes in SS were compared with Wilcoxon's Signed-Rank test. The Kaplan-Meier method was used to generate survival curves and log-rank test was used to assess differences in survival. The prognostic evaluation was also conducted in different rSS cutoffs (0, 8, and 12 points), defined in previous literature.^{12,20,21} Receiver operating characteristic (ROC) curves were constructed to assess the predictive power of bSS and rSS for 90-day mortality. The association with mortality was assessed for relevant control variables (age, gender, hypertension, diabetes, coronary artery disease, previous myocardial infarction, previous PCI, chronic heart failure, resuscitation, systolic blood pressure, left ventricular ejection fraction (LVEF), eGFR, hs-TnT, NT-proBNP, arterial blood lactate, pH, one-vessel disease, three-vessel disease, left main disease, chronic total occlusion (CTO), acute thrombus, multivessel PCI, drug-eluting stent, prior PCI TIMI flow 0, and post-PCI TIMI flow 3) by the univariate Cox regression and variables with P -value less than 0.05 were selected for multivariable analysis. The final multivariable model included age, LVEF, arterial blood lactate, eGFR, NT-proBNP, and post-PCI TIMI flow 3 and the model was built in a forward stepwise method with the Cox proportional analysis. In addition, three multivariable models were created to evaluate the additive prognostic value of bSS and rSS over clinical risk scores: bSS or rSS and either CardShock risk score,¹⁵ IABP-SHOCK II risk score¹⁶ or GRACE risk score.¹⁷ An

interaction analysis between CS timing (before or after revascularization) and rSS was also conducted. A two-tailed *P*-value of less than 0.05 was considered statistically significant. Statistical analyses were performed using SPSS 24.0.0.0 statistical software (IBM Corp, Armonk, NY, USA).

3 | RESULTS

3.1 | Baseline characteristics and SS results

The patient selection process (*n* = 61) for this substudy is described in Figure 1. Briefly, mean age was 67 (\pm 12) years, 85% were male. Median bSS1 at baseline was 22 (15–32). After wiring or thrombectomy, median bSS2 was 19 (11–30), significantly lower than bSS1 (*P* < 0.01). After revascularization, median rSS was 7 (0–13), lower than bSS1 and bSS2 (both *P* < 0.01). Only two patients had second angiography after primary PCI, but the angiogram for second procedure was available for assessment for one patient only.

Table 1 summarizes baseline characteristics according to bSS tertiles (the first tertile: bSS1 \leq 18; the second tertile: bSS1 19–27; the third tertile: bSS1 > 27). There were no differences among the tertiles except for history of previous PCI that was lower in the first tertile in comparison to higher tertiles (both *P* < 0.01). There were no differences in hemodynamic parameters and routine laboratory measurements (Table 1).

3.2 | Angiographic findings

One-third of the patients had one-vessel disease, 39% had two-vessel disease, and 23% three-vessel disease. In the first and the second bSS tertiles, only one patient (5%) in each group had CTO, while in the third tertile 75% had CTO (both *P* < 0.01). Over half of the patients (64%) had acute occlusive thrombosis of IRA and there were no differences between tertiles (*P* = 0.45). Left main was affected approximately in 15% in all tertiles (*P* = 0.99) (Table 1).

Multivessel PCI was performed with similar frequency in all bSS tertiles (28%) and the use of drug-eluting stents (33%) did not differ between the groups. Complete revascularization (rSS 0) was achieved in 28% of the patients. rSS was lower in the first and the second tertiles in comparison with the third tertile (both *P* < 0.01). More than

half of the patients (*n* = 36, 59%) had shock before PCI (Table 1) but there was no difference in either SS whether the patient had shock before or after the procedure (Supporting Information Table 1). At discharge, 94% of the patients were on dual antiplatelet therapy (Table 1).

3.3 | Mortality

All-cause 90-day mortality was 43%. Mortality rate was lower in the first tertile (19%), in comparison with the second (50%) and the third tertiles (60%) (Figure 2A). In univariate analysis, bSS predicted 90-day mortality (HR 1.04, 95% CI 1.01–1.08) and the association was independent after adjustment for control variables (HR 1.06, 95% CI 1.01–1.10). bSS predicted mortality also after adjustment with the IABP-SHOCK II (HR 1.05, 95% CI 1.01–1.09) and GRACE (HR 1.04, 95% CI 1.00–1.08) risk scores but not when adjusted for the Card-Shock risk score (HR 1.02, 95% CI 0.98–1.06) (Table 2A).

There were no statistically significant differences in mortality when using different rSS cutoff points (0, 8, and 12) (Figure 2B,D). In univariate analysis, rSS as a continuous variable correlated with 90-day mortality (HR 1.04, 95% CI 1.00–1.07), but not independently in any of the multivariable models (Table 2A). rSS as a categorical variable did not associate with mortality in univariate or multivariable analysis (Table 3A). There was no interaction between the timing of CS (before or after coronary angiography) and rSS. Figure 3 depicts the ROC-curves and AUCs for the bSS and rSS in comparison with the three risk scores.

4 | DISCUSSION

To the best of our knowledge, this is the first study that evaluates the prognostic value of bSS and rSS in patients with STEMI-related CS. We found distinctive improvement from bSS to rSS implying successful revascularization. bSS seems to associate with short-term mortality, but the additive value over pre-existing clinical risk scores is limited. The prognostic effect of rSS could not be shown, but post-PCI TIMI flow 3 of IRA predicted better survival.

The median bSS in this study was lower than in the original SYN-TAX study,²² but higher than in previous studies concerning STEMI.^{2,5,6,12} The median bSS was higher than described in another CS population,²³ yet the definition of CS in our study was stricter. Most of the patients in this study had multivessel disease and one fourth had CTO, indicating that the patients with STEMI-related CS have pre-existing complex coronary artery lesions. Regarding the risk of CS in STEMI patients, CS has been more prevalent in the patients with high SS^{5,6} and SS has been identified as an independent predictor of CS in ACS patients.²³ The results are in line with our finding of high bSS in STEMI-related CS.

The prognostic value of bSS in STEMI-related CS was evaluated for the first time. bSS retained prognostic value in CS but its clinical relevance was limited, especially when added to recently developed risk scores for CS. The result differs to some extent from observations in non-shock STEMI patients in which bSS was credited with a robust predictive value.^{2,4–6,10} However, in these STEMI populations, the

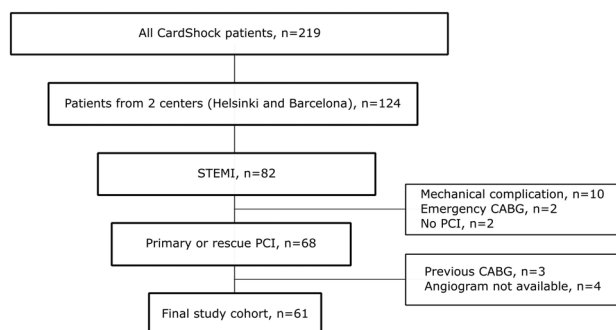


FIGURE 1 Patient flowchart—STEMI patients treated with primary PCI or rescue PCI were included in this substudy. Exclusion criteria were previous CABG, emergency CABG after angiography, or mechanical complication of STEMI as an etiology of CS

TABLE 1 Baseline characteristics, SYNTAX scores, clinical data, and mortality in bSS1 tertiles

	First tertile bSS1 ≤ 18 n = 21	Second tertile bSS1 19–27 n = 20	Third tertile bSS > 27 n = 20	P-value
Age, years (SD)	62 (±12)	68 (±13)	70 (±12)	0.92
Male gender, n (%)	20 (95%)	14 (70%)	18 (90%)	0.06
Resuscitation, n (%)	9 (43%)	7 (35%)	3 (15%)	0.12
<i>SYNTAX scores</i>				
bSS2, pts (IQR)	7 (4–11)	13 (12–16)	32 (22–36)	<0.01
rSS, pts (IQR)	0 (0–5)	6 (1.0–10)	20 (10–28)	<0.01
<i>Medical history</i>				
Coronary artery disease, n (%)	2 (10%)	4 (20%)	7 (35%)	0.13
Previous PCI, n (%)	0 (0%)	3 (15%)	6 (30%)	<0.01
Diabetes, n (%)	5 (24%)	2 (10%)	8 (40%)	0.08
Hypertension, n (%)	11 (52%)	11 (55%)	13 (65%)	0.69
Hypercholesterolemia, n (%)	10 (48%)	11 (55%)	9 (45%)	0.81
Smoking, n (%)	13 (62%)	14 (70%)	8 (42%)	0.08
<i>Clinical parameters</i>				
LVEF, % (SD)	37 (±15)	34 (±14)	29 (±9)	0.17
SBP, mmHg (SD)	77 (±23)	77 (±11)	76 (±12)	0.98
Serum lactate, mmol/l (IQR)	2.2 (1.2–3.2)	2.3 (1.7–6.3)	2.8 (2.1–4.4)	0.31
Creatinine, μmol/l (IQR)	89 (72–110)	97 (74–116)	112 (85–136)	0.20
eGFR, ml/min (IQR)	88 (65–104)	78 (42–98)	67 (41–87)	0.15
Hs-TnT, ng/l (IQR)	2,427 (849–6,810)	7,236 (3,677–11,943)	2,889 (1,828–8,965)	0.04
NT-proBNP, ng/l (IQR)	198 (133–942)	1,471 (253–3,977)	3,914 (481–16,551)	<0.01
<i>Angiographic data</i>				
Acute occlusive thrombosis, n (%)	12 (57%)	15 (75%)	12 (60%)	0.45
Chronic total occlusion, n (%)	1 (4.8%)	1 (5.0%)	15 (75%)	<0.01
One-vessel disease, n (%)	14 (67%)	8 (42%)	0 (0%)	<0.01
Three-vessel disease, n (%)	1 (4.8%)	2 (11%)	11 (55%)	<0.01
Multivessel disease, n (%)	7 (33%)	11 (55%)	20 (100%)	<0.01
Left main disease, n (%)	3 (14%)	3 (16%)	3 (15%)	0.99
CS before angiography, n (%)	12 (57%)	13 (65%)	11 (55%)	0.79
Time from shock to angiography, min (IQR)	68 (23–112)	40 (15–86)	75 (15–90)	0.57
Time from angiography to shock, min (IQR)	180 (60–300)	20 (15–75)	98 (45–175)	0.47
Second PCI, n (%)	0 (0%)	1 (5.0%)	1 (5%)	0.41
<i>Treatment</i>				
Multivessel PCI, n (%)	4 (19%)	6 (30%)	7 (35%)	0.50
Drug-eluting stent, n (%)	8 (38%)	6 (30%)	6 (30%)	0.82
Complete revascularization (rSS 0), n (%)	12 (57%)	5 (25%)	0 (0%)	<0.01
Post-PCI TIMI 3, n (%)	14 (67%)	12 (60%)	14 (74%)	0.66
IABP, n (%)	10 (48%)	14 (70%)	13 (65%)	0.31
<i>Medication at discharge^a</i>				
Aspirin ^a , n (%)	16 (94%)	11 (100%)	8 (100%)	0.47
Clopidogrel/prasugrel ^a , n (%)	15 (88%)	11 (100%)	7 (88%)	0.22
<i>Endpoints</i>				
90-Day mortality, n (%)	4 (19%)	10 (50%)	12 (60%)	0.02

Abbreviations: bSS, baseline SYNTAX Score; CS, cardiogenic shock; eGFR, estimated glomerular filtration; hs-TnT, high-sensitivity troponin T; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; MACCE, major adverse cardiac and cerebrovascular events; NT-proBNP, N-terminal pro-b-type natriuretic peptide; PCI, percutaneous coronary intervention; rSS, residual SYNTAX Score; SBP, systolic blood pressure. Data are presented as numbers (%), means (SD) and medians (IQR).

^a Percentages calculated in patients who were discharged (n = 36).

prevalence of CS was very low, which could explain the difference in outcomes. In the present study, bSS had some additive predictive value over the GRACE and IABP-SHOCK II risk scores. Previously, in a non-CS STEMI population, bSS was shown to be useful in risk

stratification over GRACE risk score.²⁴ Nevertheless, we believe, that in the acute clinical context of CS, using clinical risk scores for risk stratification is more feasible and accurate than assessing the exact burden of coronary artery disease with bSS. However, the sample size

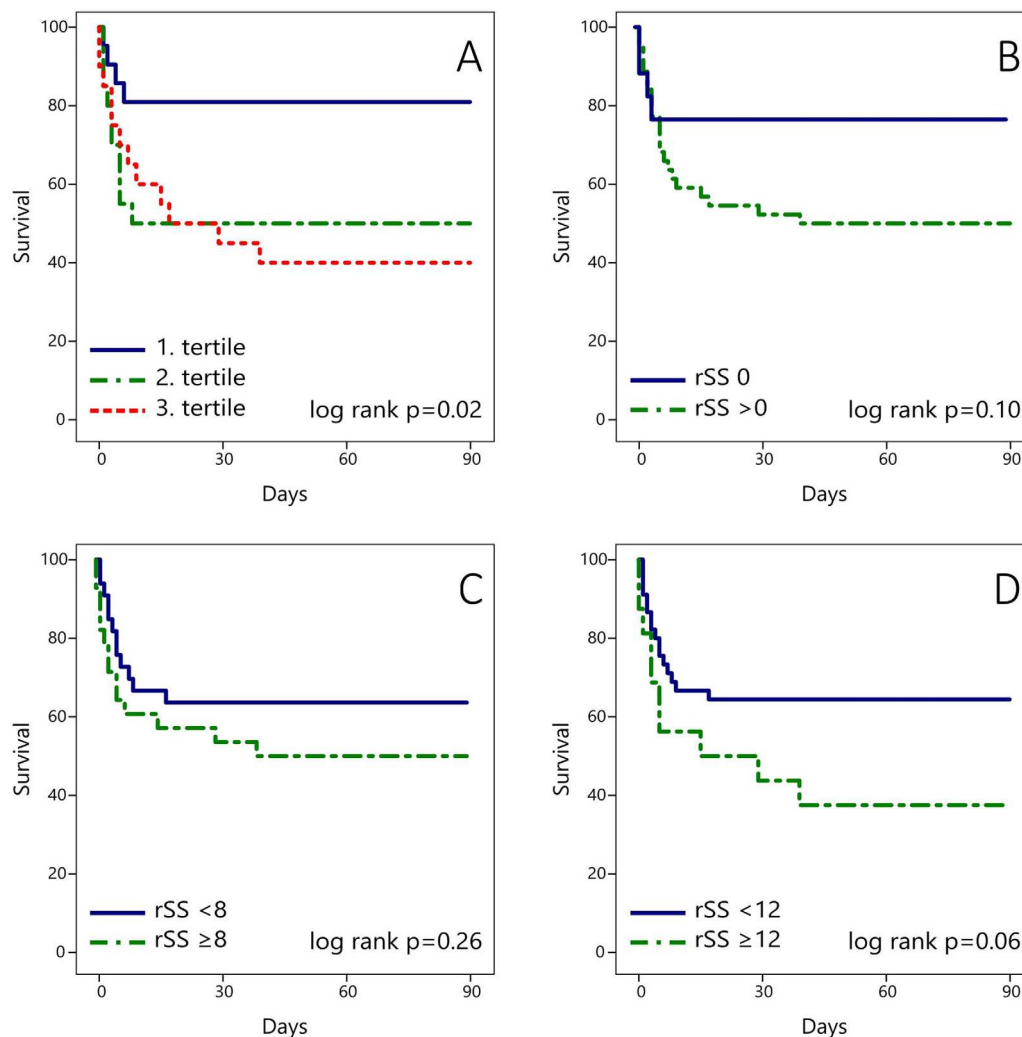


FIGURE 2 Ninety-day survival analysis—A, Kaplan–Meier’s survival curves in bSS1 tertiles; B–D, and in different rSS cutoff points [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 2 The Cox survival models with bSS and rSS as continuous variable for 90-day all-cause mortality

(A)		HR	95% CI	P-value		HR	95% CI	P-value
Univariable	bSS ^b	1.04	1.01–1.08	0.02	rSS ^b	1.04	1.00–1.08	0.04
Multivariable								
Control variables ^a								
	bSS ^b	1.06	1.01–1.10	0.03	rSS ^b	1.03	0.99–1.08	0.14
	LVEF ^c	1.80	1.22–2.65	<0.01	LVEF ^c	1.87	1.31–2.69	<0.01
	Lactate ^d	1.23	1.08–1.39	<0.01	Lactate ^d	1.21	1.08–1.36	<0.01
	Post-PCI TIMI 3	0.26	0.10–0.65	<0.01	Post-PCI TIMI 3	0.28	0.11–0.67	<0.01
Clinical risk scores								
	CardShock ^b	1.79	1.36–2.34	<0.01	CardShock ^b	1.81	1.38–2.37	<0.01
	bSS ^b	1.02	0.98–1.06	0.29	rSS ^b	1.02	0.98–1.06	0.44
	IAPB-SHOCK II ^b	2.00	1.52–2.62	<0.01	IAPB-SHOCK II ^b	1.86	1.42–2.42	<0.01
	bSS ^b	1.05	1.01–1.09	<0.01	rSS ^b	1.03	0.99–1.07	0.08
	GRACE ^b	1.02	1.00–1.03	0.01	GRACE ^b	1.02	1.00–1.03	0.01
	bSS ^b	1.04	1.00–1.08	0.04	rSS ^b	1.03	0.99–1.07	0.11

^a See Section 2 for variables assessed.

^b Per one point increase.

^c Per 10% decrease.

^d Per 1 mmol/l increase.

TABLE 3 The Cox survival models with rSS as categorical variable with different cutoffs for 90-day all-cause mortality

		HR	95% CI	P-value
(A)				
Univariable	rSS > 0	2.34	0.81–6.80	0.09
	rSS ≥ 8	1.55	0.72–3.35	0.27
	rSS ≥ 12	2.05	0.93–4.52	0.08
Multivariable				
Control variables ^a	LVEF ^b			<0.01 ^e
	Lactate ^c			<0.01 ^e
	Post-PCI TIMI 3			<0.01 ^e
and either	(1) rSS > 0	2.46	0.83–7.29	0.11
	(2) rSS ≥ 8	1.27	0.55–2.94	0.58
	(3) rSS ≥ 12	1.54	0.65–3.66	0.33
Clinical risk scores				
and either	CardShock ^d			<0.01 ^e
	(1) rSS > 0	2.41	0.80–7.26	0.12
	(2) rSS ≥ 8	1.05	0.48–2.31	0.91
	(3) rSS ≥ 12	1.26	0.55–2.88	0.58
	IABP-SHOCK II ^d			<0.01 ^e
	(1) rSS > 0	2.05	0.70–6.00	0.19
	(2) rSS ≥ 8	1.36	0.62–2.99	0.45
	(3) rSS ≥ 12	1.53	0.67–3.49	0.31
	GRACE ^d			0.01 ^e
	(1) rSS > 0	1.83	0.62–5.43	0.28
	(2) rSS ≥ 8	1.44	0.67–3.11	0.36
	(3) rSS ≥ 12	1.71	0.77–3.80	0.19

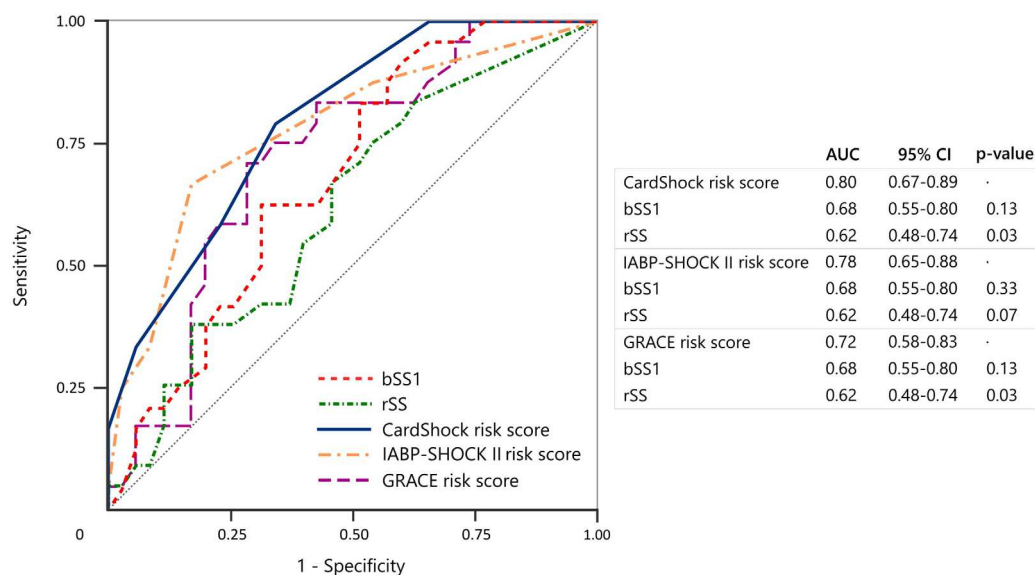
^a See Section 2 for variables assessed.^b Per 10% decrease.^c Per one mmol/l increase.^d Per one point increase.^e P-values calculated and valid for all categorical rSS multivariable models.

in this study is quite small, meaning that the mortality analyses do have to be assessed with caution.

Regarding the analysis of rSS, some of the patients were diagnosed with CS only after coronary angiography, when optimally, all patients should have presented with CS before revascularization. However, this study reflects real life practice, since CS most often develops during hospitalization,²⁵ and in the current era of immediate revascularization in STEMI, it is evident that some patients develop CS only after the procedure. In addition, most study patients who developed CS after revascularization, did it during the first hours after angiography, suggesting that the pathophysiologic changes of CS had probably started even before coronary angiography, and some clinical signs of CS may already have been present. Therefore, we believe our results can be useful in the clinical practice.

The distinctive improvement from bSS to rSS implies successful revascularization. However, the rate of complete revascularization (rSS 0) was lower than in prior studies^{13,20,21,26} and it was reached most often in patients with the lowest bSS. As complete revascularization is not always achievable in acute setting, previous studies have examined the concept of reasonable incomplete revascularization in ACS, but not in CS. Previously, the cutoff has been most often set at rSS 8,^{14,20,21,27} or 12 points.^{9,12} In contrast to the earlier studies in non-CS populations, we found no prognostic effect whether we used the rSS cutoff of 8 or 12 points.

We could not demonstrate any predictive value of rSS in STEMI-related CS. In previous studies, rSS independently predicted mortality in STEMI.^{12,14,27} Recent CULPRIT-SHOCK trial showed worse survival in CS in patients treated with multivessel PCI in comparison with revascularization of culprit artery only,⁸ and the earlier studies have shown similar results²⁸ or they have found no survival benefit.²⁹ In our study, rSS had only modest association with mortality in univariate analysis, but the prognostic value was outweighed by the clinical variables and the risk scores in adjusted analysis. The difference in the predictive power of rSS probably relates to the difference between

**FIGURE 3** The prognostic ability of SYNTAX scores—ROC curves of bSS, rSS, and clinical risk scores [Color figure can be viewed at wileyonlinelibrary.com]

the severity of clinical condition, i.e., CS and any ACS. The patients in our study were seriously ill: one third of the patients had been resuscitated and over half had CS before PCI. In such circumstances, revascularization is challenging, and treatment targets must include hemodynamic stability, adequate organ perfusion and limiting multi-organ failure. Regarding the timing of CS and the completeness of revascularization, we found that rSS was equal in patients who developed CS before or after angiography. This finding could suggest that less complete revascularization was not the cause of CS in the patients who developed CS after the procedure. However, considering the small sample size of the study, straightforward conclusions are difficult to make regarding the relationship between the failure of revascularization and the cause of the CS. Nevertheless, in line with previous studies, successful revascularization of the IRA, defined as achieving post-PCI TIMI flow 3, was associated with lower mortality.¹⁶ This may suggest that restoring the blood flow to the IRA is more important than targeting complete revascularization and low rSS.

5 | LIMITATIONS

Several limitations of our study need to be considered. First, the cohort consists of a limited number of patients, and despite high mortality rates, the number of events in mortality analyses is small. Nevertheless, this is to our knowledge the first, and thus the largest analysis of SS in a cohort of CS patients. Second, interpretation of the angiograms was not centralized, but the SS was analyzed in each hospital by one local experienced cardiologist. However, both were well trained to calculate the SS. Third, the CardShock risk score was developed from the original CardShock cohort including also the patients in the current study, which could affect the analysis of the prognostic value of bSS and rSS over the CardShock risk score. However, the added value of SS was limited also when comparing to two other risk scores.

6 | CONCLUSION

In STEMI-related CS, the additive value of bSS and rSS over clinical assessment and risk scores is limited. Our results support that in STEMI-related CS, restoring the blood flow to the IRA is essential, and more important than opting for the complete revascularization with PCI.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

ORCID

Tuija Javanainen  <https://orcid.org/0000-0003-2764-7253>

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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